

# Fauci's Testimony on Capitol Hill Was That of a Guilty Man

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By Peter A. McCullough, MD, MPH

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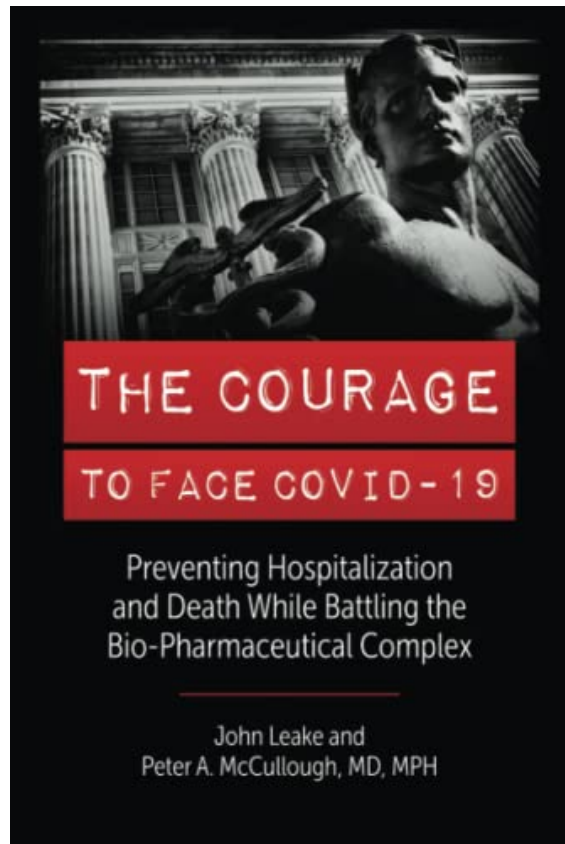
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I testified along with [Drs. Ryan Cole, and Kirk Milhoan, before a US Congressional Panel on January 12, 2024](#). This followed two days of inquiry former NIAID Director, Dr. Anthony Fauci by the US House of Representatives **Select** Subcommittee on the **Coronavirus Pandemic**. Reports indicate that [Fauci was evasive and over one hundred times said "I dont know" or "I dont recall."](#)

[THE COURAGE TO FACE CO...](#) McCullough MD, Peter A. Best Price: \$6.50 Buy New \$10.24 (as of 03:00 UTC - Details) I told Corri and Allan Hunsberger of [Talk Truth](#) on January 19, 2024, that during our testimony as treating physicians who have faced the pandemic full-on, both with ambulatory management of SARS-CoV-2 illness and now serious adverse events caused by COVID-19 vaccines, we did not evade questions and answered every query from the panel with honesty and integrity.



What is reported about Fauci's testimony suggests he is a guilty man. A witness, subject, or target who is truly culpable for an atrocity will be evasive and do everything to conceal participation in what has occurred. It is likely when all the evidence is fully considered that the following will be recognized:

Fauci likely will be shown to have participated in the design and creation of the gain-of-function chimeric virus SARS-CoV-2 with co-conspirators Dr. Ralph Baric at the University of North Carolina, Chapel Hill, (and his team), Dr. Peter Daszak leader of the EcoHealth Alliance,

and Chinese virologist Dr. Shi Zhengli with her research group at the Wuhan Institute of Virology. Likely final conclusions will be:



1. Fauci conspired with Baric to withhold the genetic code from Baric's virus (SARS-like WIV-1 CoV) published in 2015-2016 because it closely matched the “ancestral” Wuhan strain of SARS-CoV-2 that infected the world.

## A SARS-like cluster of circulating bat coronaviruses shows potential for human emergence

Vincent D. Menachery<sup>1</sup>, Boyd L. Yount Jr.<sup>2</sup>, Amy C. Sims<sup>3</sup>, Kari Debbink<sup>1,2</sup>, Sudhakar Agrihotharam<sup>1</sup>, Lisa E. Gralinski<sup>1</sup>, Jessica A. Plante<sup>1</sup>, Rachel L. Graham<sup>1</sup>, Trevor Scobey<sup>1</sup>, Xing-Yi Ge<sup>4</sup>, Eric F. Davidson<sup>1</sup>, Scott H. Randell<sup>5</sup>, Antonio Lanzavecchia<sup>6</sup>, Wayne A. Marasco<sup>6,7</sup>, Zhong-Li Shi<sup>8</sup> & Ralph S. Baric<sup>1,2</sup>

The emergence of severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome (MERS-CoV) underscores the threat of cross-species transmission events leading to outbreaks in humans. Here we examine the disease potential of a SARS-like virus, SHC014-CoV, which is currently circulating in Chinese horseshoe bat populations<sup>1</sup>. Using the SARS-CoV reverse genetics system<sup>2</sup>, we generated and characterized a chimeric virus expressing the spike of bat coronavirus SHC014 in a mouse-adapted SARS-CoV backbone. The results indicate that group 2b viruses encoding the SHC014 spike in a wild-type backbone can efficiently use multiple orthologs of the SARS receptor human angiotensin converting enzyme II (ACE2), replicate efficiently in primary human airway cells and achieve *in vitro* titres equivalent to epidemic strains of SARS-CoV. Additionally, *in vivo* experiments demonstrate replication of the chimeric virus in mouse lung with notable pathogenesis. Evaluation of available SARS-based immune therapeutic and prophylactic modalities revealed poor efficacy; both monoclonal antibody and vaccine approaches failed to neutralize and protect from infection with CoVs using the novel spike protein. On the basis of these findings, we synthetically re-derived an infectious full-length SHC014 recombinant virus and demonstrate robust viral replication both *in vitro* and *in vivo*. Our work suggests a potential risk of SARS-CoV re-emergence from viruses currently circulating in bat populations.

the affected regions<sup>3</sup>. Although public health measures were able to stop the SARS-CoV outbreak<sup>4</sup>, recent metagenomics studies have identified sequences of closely related SARS-like viruses circulating in Chinese bat populations that may pose a future threat<sup>1,5</sup>. However, sequence data alone provides minimal insights to identify and prepare for future zoonotic viruses. Therefore, to examine the emergence potential (that is, the potential to infect humans) of circulating bat CoVs, we built a chimeric virus encoding a novel, zoonotic CoV spike protein—from the bat SHC014-CoV sequence that was isolated from Chinese horseshoe bats<sup>1</sup>—in the context of the SARS-CoV mouse-adapted backbone. The hybrid virus allowed us to evaluate the ability of the novel spike protein to cause disease independently of other necessary adaptive mutations in its natural backbone. Using this approach, we characterized CoV infection mediated by the SHC014 spike protein in primary human airway cells and *in vivo*, and tested the efficacy of available immune therapeutics against SHC014-CoV. Together, the strategy translates metagenomics data to help predict and prepare for future emergent viruses.

The sequences of SHC014 and the related batWIV1-CoV show that these CoVs are the closest relatives to the epidemic SARS-CoV strains (Fig. 1a,b); however, there are important differences in the 14 residues that are critical for host range: Y448, L472, M479, T487 and T491 (Sup. Fig. 1). None of these residues vary from the epidemic SARS-CoV Urbani strain, but they were not expected to alter binding to ACE2 (Supplementary Fig. 1a,b and Supplementary Table 1). This data is consistent with both neutralization experiments that show

## SARS-like WIV1-CoV poised for human emergence

Vincent D. Menachery<sup>1</sup>, Boyd L. Yount Jr.<sup>2</sup>, Amy C. Sims<sup>3</sup>, Kari Debbink<sup>1,2</sup>, Sudhakar S. Agrihotharam<sup>1</sup>, Lisa E. Gralinski<sup>1</sup>, Rachel L. Graham<sup>1</sup>, Trevor Scobey<sup>1</sup>, Jessica A. Plante<sup>1</sup>, Scott H. Randell<sup>5</sup>, Jessica Swanson<sup>6</sup>, Timothy P. Sheahan<sup>6</sup>, Raymond J. Pickles<sup>6,7</sup>, Davide Corti<sup>8,9</sup>, Scott H. Randell<sup>5</sup>, Antonio Lanzavecchia<sup>6</sup>, Wayne A. Marasco<sup>6</sup>, and Ralph S. Baric<sup>1,2</sup>

<sup>1</sup>Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, <sup>2</sup>Department of Microbiology and Immunology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, <sup>3</sup>Division of Microbiology, National Center for Toxicological Research, Food and Drug Administration, Jefferson, AR 72079, <sup>4</sup>Department of Cell Biology and Physiology and Marlow-Lung Institute/Cytic Fibrosis Center, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, <sup>5</sup>Institute for Research in Biomedicine, Bellinzona, Switzerland, <sup>6</sup>Institute of Microbiology, Eidgenössische Technische Hochschule Zurich, Zurich, Switzerland, <sup>7</sup>Immuno-Biotech SA, Bellinzona, Switzerland, and <sup>8</sup>Department of Microbiology and AIDS, Dana-Farber Cancer Institute/Department of Medicine, Harvard Medical School, Boston, MA 02115

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Outbreaks from zoonotic sources represent a threat to both human disease as well as the global economy. Despite a wealth of metagenomics studies, methods to leverage these datasets to identify future threats are underdeveloped. In this study, we describe an approach that combines existing metagenomics data with reverse genetics to engineer reagents to evaluate emergence and pathogenic potential of circulating zoonotic viruses. Focusing on the severe acute respiratory syndrome (SARS)-like viruses, the results indicate that the WIV1-coronavirus (CoV) cluster has the ability to directly infect and may undergo limited transmission in human populations. However, *in vivo* attenuation suggests additional adaptation is required for epidemic disease. Importantly, available SARS monoclonal antibodies offered success in limiting viral infection absent from available vaccine approaches. Together, the data highlight the utility of a platform to identify and prioritize zoonotic strains harbored in animal reservoirs and document the threat posed by WIV1-CoV for emergence in human populations.

SARS | CoV | emergence | Spike | WIV1

strategies against SARS were effective against WIV1-CoV spike unlike available vaccine approaches. Together, the results highlight the utility of developing platforms to evaluate circulating zoonotic viruses as threats for future emergence and epidemic potential.

### Results

The discovery of SARS-like virus clusters that bridge the gap between the epidemic strains and related precursor CoV strain HKU1 virus provided the best evidence for emergence of SARS-CoV from Chinese horseshoe bats (5). Comparing the receptor binding domain (RBD), SARS-CoV Urbani and WIV1 share homology at 11 of the 14 contact residues with human ACE2 (Fig. 1a); importantly, the three amino acid changes represent relatively conservative substitutions not predicted to ablate binding (Fig. 1b). Therefore, exploring WIV1 strains allows examination of emergence, pathogenesis potential, and adaptation requirements. Using the SARS-CoV infectious clone as a template (7), we designed and synthesized a full-length infectious clone of WIV1-CoV consisting of six plasmids that could be

2. Menachery et al manuscripts describing the creation of SARS-CoV-2 prototype, declaring gain-of-function methods used, indicating NIH NIAID funding and approval, and thanks given to the EcoHealth Alliance and the Wuhan Institute of Virology.

## Read the Whole Article

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